R^{45} is selected from -Y"-R^{19};

Y" is selected from a chemical bond, O, NR^0-, and a hydrocarbon chain having from 1 to 4 carbon atoms, and optionally substituted with one or more of halo, alkyl, cycloalkyl, alkenyl, alkynyl, aralkyl, CO_2R^0, $C(O)R^0$, $C(O)N(R^0)_2$, CN, CF_3, $N(R^0)_2$, NO_2, and OR^0;

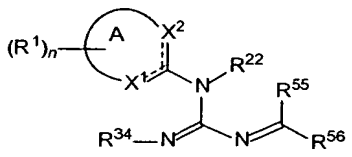
R^{19} is selected from the group consisting of H, alkyl, cycloalkyl, alkenyl, alkynyl, aralkyl, CF_3, aryl, and a heterocyclic ring; and

each R^0 is independently selected from H, alkyl, cycloalkyl, aralkyl, aryl and a heterocyclic ring.

24. The method of claim 23 wherein X^1 is N.

25. The method of claim 24 wherein X^2 is N.

26. The method of claim 1, comprising administering a therapeutically effective amount of a compound having the formula V



(V)

wherein:

ring A is a 5-, 6-, or 7- membered ring or a 7- to 12-membered fused bicyclic ring;

X^1 is selected from N, N-R^0 or C-R^1;

X^2 is selected from N, N-R^0 or C-R^1;

the dotted lines represent optional double bonds;

each R^1 is independently selected from the group consisting of H, alkyl, cycloalkyl, alkenyl, alkynyl, aralkyl, CN, CF_3, NO_2, OR^{11}, $-(CH_2)_pC(O)(CH_2)_qR^{11}$, $-(CH_2)_pC(O)N(R^{12})(R^{13})$, $-(CH_2)_pC(O)O(CH_2)_qR^{11}$, $-(CH_2)_pN(R^{11})C(O)R^{11}$, $-(CH_2)_pN(R^{12})(R^{13})$, $-N(R^{11})SO_2R^{11}$, $-OC(O)N(R^{12})(R^{13})$, $-SO_2N(R^{12})(R^{13})$, halo, aryl, and a heterocyclic ring, and additionally or alternatively, two R^1 groups on adjacent ring atoms form a 5- or 6-membered fused ring which contains from 0 to 3 heteroatoms;

n is 0 to 6,

each R^{11} is independently selected from H, alkyl, cycloalkyl, alkenyl, alkynyl, aralkyl, aryl, and a heterocyclic ring;

each R^{12} and R^{13} are independently selected from H, alkyl, cycloalkyl, alkenyl, alkynyl, aralkyl, aryl, and a heterocyclic ring; or R^{12} and R^{13} may be taken

together with the nitrogen to which they are attached form a 5- to 7- membered ring which may optionally contain a further heteroatom;

p is 0 to 4;

q is 0 to 4;

R^{22} is selected from H and C_{1-3} alkyl;

R^{34} is selected from H, NO_2, CN, alkyl, cycloalkyl, alkenyl, alkynyl, aralkyl, aryl and a heterocyclic ring;

R^{55} is selected from H, alkyl, cycloalkyl, alkenyl, alkynyl, aralkyl, aryl, and a heterocyclic ring;

R^{56} is selected from -Y"-R^{19};

Y" is selected from a chemical bond, O, NR^0-, and a hydrocarbon chain having from 1 to 4 carbon atoms, and optionally substituted with one or more of halo, alkyl, cycloalkyl, alkenyl, alkynyl, aralkyl, CO_2R^0, $C(O)R^0$, $C(O)N(R^0)_2$, CN, CF_3, $N(R^0)_2$, NO_2, and OR^0;

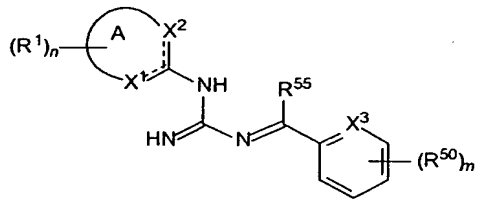
R^{19} is selected from the group consisting of H, alkyl, cycloalkyl, alkenyl, alkynyl, aralkyl, CF_3, aryl, and a heterocyclic ring; and

each R^0 is independently selected from H, alkyl, cycloalkyl, aralkyl, aryl and a heterocyclic ring.

27. The method of claim 26 wherein X^1 is N.

28. The method of claim 27 wherein X^2 is N.

29. The method of claim 1, comprising administering a therapeutically effective amount of a compound having the formula V_a



(V_a)

wherein:

ring A is a 5-, 6-, or 7- membered ring or a 7- to 12-membered fused bicyclic ring;

X^1 is selected from N, N-R^0 or C-R^1;

each R^0 is independently selected from H, alkyl, cycloalkyl, aralkyl, aryl and a heterocyclic ring;

h is 0 to 4;

i is 0 to 4; and

j is 0 to 4.

52. A method for determining whether a substance is an inhibitor or an activator of a theramutein which is capable of eliciting a detectable phenoresponse, which comprises:

a) incubating a first cell which expresses the theramutein at a substantially constant level with the substance;

b) incubating a second cell which expresses a corresponding prototheramutein at a substantially constant level with a known inhibitor or activator of the prototheramutein;

c) comparing a phenoresponse of the second cell to the known inhibitor or activator of the prototheramutein to the phenoresponse of the first cell to the substance; and

d) determining that the phenoresponse of the first cell is inhibited or activated to at least the same degree as the phenoresponse of the second cell is inhibited or activated by the known inhibitor or activator of the prototheramutein, thereby identifying the substance as an inhibitor or an activator of the theramutein.

53. The method of Claim 52, wherein the phenoresponse of the cell expressing the theramutein to the substance is greater than the phenoresponse of the cell expressing the prototheramutein to the known inhibitor or activator of the theramutein.

54. A method for determining whether a substance is a specific inhibitor or specific activator of a theramutein, which comprises:

a) providing a test cell which expresses the theramutein and which gives rise to a detectable phenoresponse;

b) treating the test cell with the substance;

c) examining the treated cell to determine whether the phenoresponse is modulated by treatment with the substance.

55. The method of Claim 52 or 54, wherein the theramutein or prototheramutein is a component of a signal transduction cascade.

56. The method of Claim 52 or 54, wherein the theramutein or prototheramutein is an enzyme.

57. The method of Claim 52 or 54, wherein the theramutein or prototheramutein is a protein kinase.

58. The method of Claim 52 or 54, wherein the theramutein or prototheramutein is a tyrosine kinase.

59. The method of Claim 52 or 54, wherein the theramutein or prototheramutein is a receptor tyrosine kinase.

60. The method of Claim 52 or 54, wherein the or prototheramutein is $p210^{Bcr\text{-}Abl}$.

61. The method of Claim 52 or 54, wherein the or prototheramutein is the T315I mutant of $p210^{Bcr\text{-}Abl}$.

62. The method of Claim 52 or 54, wherein the phenoresponse is a change in a cultural, morphological, or transient characteristic of the cell.

63. The method of Claim 52 or 54, wherein the phenoresponse includes phosphorylation of an intracellular substrate of the theramutein.

64. The method of Claim 52 or 54, wherein the phenoresponse is detected on a subcellular fraction of the cell.